A COMPARATIVE STUDY OF ANAESTHETIC EFFICACY OF INTRATHECAL ISOBARIC 0.5% ROPIVACAINE AND 0.5%BUPIVACAINE IN LOWER ABDOMINAL SURGERIES

DISSERTATION SUBMITTED FOR M.D .DEGREE EXAMINATION

BRANCH X –ANAESTHESIOLOGY

THANJAVUR MEDICAL COLLEGE

THE TAMILNADU DR.MGR MEDICAL

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BONAFIDE CERTIFICATE

This is to certify that dissertation entitled ***'A comparative study of Anaesthetic efficacy of intrathecal isobaric 0.5% Ropivacaine and 0.5% Bupivacaine in lower abdominal surgeries'*** *is a bonafide record of the work done by* ***Dr.H.Ganga Nagalakshmi*** *under my supervision and guidance in the Dept of Anaesthesialogy,at Thanjavur Medical College Thanjavur during the period of her postgraduate study from May 2008 to April 2011, for the partial fullfillment of M.D (Branch x-Anaesthesialogy)Degree.*

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DECLARATION

I declare that the dissertation entitled A comparative study of anaesthetic efficacy of intrathecal isobaric 0.5% Ropivacaine and 0.5% Bupivacaine in lower abdominal surgeries has been conducted by me at the Department of Anaesthesiology, Thanjavur medical college, under the guidance and supervision of the Head of the Dept.Prof R.Muthukumaran. M.D.,D.A and it is submitted in partial fulfillment for the award of the degree of M.D branch X –anaesthesiology for April 2011 examination to be held under the Tamilnadu DR. M.G.R medical university, Chennai. This has not been submitted previously by me for the award of any degree or diploma for any other university.

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INTRODUCTION

August Bier was the first person to introduce spinal anesthesia on 16-08-1898 using 0.5 cocaine which is the first known local anesthetic. Spinal anaesthesia has a long history of success in producing profound nerve block in a large part of the body by the relatively simple injection of a small amount of local anaesthetic.

The choice of local anaesthetics is determined by the duration of surgery and by the intensity of motor blockade that is required. Lignocaine was the first amide local anaesthetic and it replaced esters following its clinical introduction in the early 1950's. Lignocaine does not have allergic sensitization, seen with esters. Lignocaine was extensively used local anaesthetic for spinal anaesthesia, but now the use has fallen dramatically due to concerns regarding transient neurological symptoms.

This prompted search for alternatives. Bupivacaine is the first long acting amide local anaesthetic. Its advantage when compared to lignocaine is its longer duration of action and differential sensory motor

block. It is due to increased lipid solubility and protein binding. But it has lower therapeutic index with respect to cardiovascular toxicity.

The increase in day care surgery has generated a need for a local anaestheic with a quick onset and shorter duration of action allowing early ambulation. Moreover, the major concern about the cardiotoxicity of bupivacaine has led to the development of ropivacaine, a new long acting amide. It is a part of the homologous series that includes bupivacaine and mepivacaine. It has an isopropyl group bound to piperdine nitrogen in place of mepivacaine's, methyl group and bupivacaine's butyl group. It is manufactured as pure s-enantiomer rather than a racemic mixture.

The L form is less cardiotoxic and has shorter duration of action than bupivacaine. Its low lipid solubility resulted in reduced risk of negative inotropism and decreased affinity for cardiac sodium channels than bupivacaine. Thus it has an improved safety profile over bupivacaine. It is available in isobaric, hyperbaric forms. The major clinical advantage of isobaric solution is that the patient's position during and after injection have no effect on the spread of local anaesthetic in cerebrospinal fluid. Thus isobaric solution do not tend to

distribute as far from the site of injection. It is useful when lower thoracic dermatomal sensory block is desired and when degree of sympathetic blockade needs to be minimized.

Hence the present study has been undertaken to compare the efficacy and hemodynamic effects of isobaric bupivacaine and isobaric ropivacaine for lower abdominal surgeries.

AIM OF THE STUDY

The purpose of this study is to compare the anesthetic efficacy of intrathecal isobaric ropivacaine 0.5% with bupivacaine 0.5% in lower abdominal surgeries with respect to

a. Onset and duration of sensory block
b. Onset, quality and duration of motor block
c. Hemodynamic changes

ANATOMY

Spinal (subarachnoid / intrathecal) anaesthesia is a form of central neuraxial block in which a temporary interruption of neural transmission is achieved following injection of local anaesthetic and/or adjuvant solutions into the subarachnoid space. Spinal anaesthesia is one of the most frequently employed techniques of regional anaesthesia.

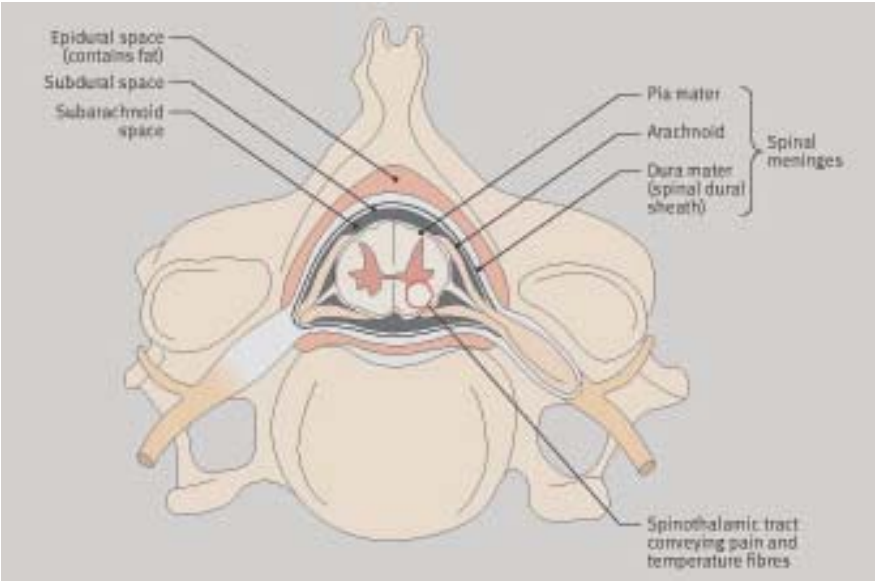
The vertebral canal extends from the foramen magnum to the sacral hiatus. It is formed by the dorsal spine, pedicles and lamina of successive vertebrae (7 cervical, 12 thoracic, 5 lumbar and 5 sacral). The vertebrae are held together by a series of overlapping ligaments namely the anterior and posterior longitudinal ligaments, ligamentum flavum, interspinous ligament, supraspinous ligament and the intervertebral discs.

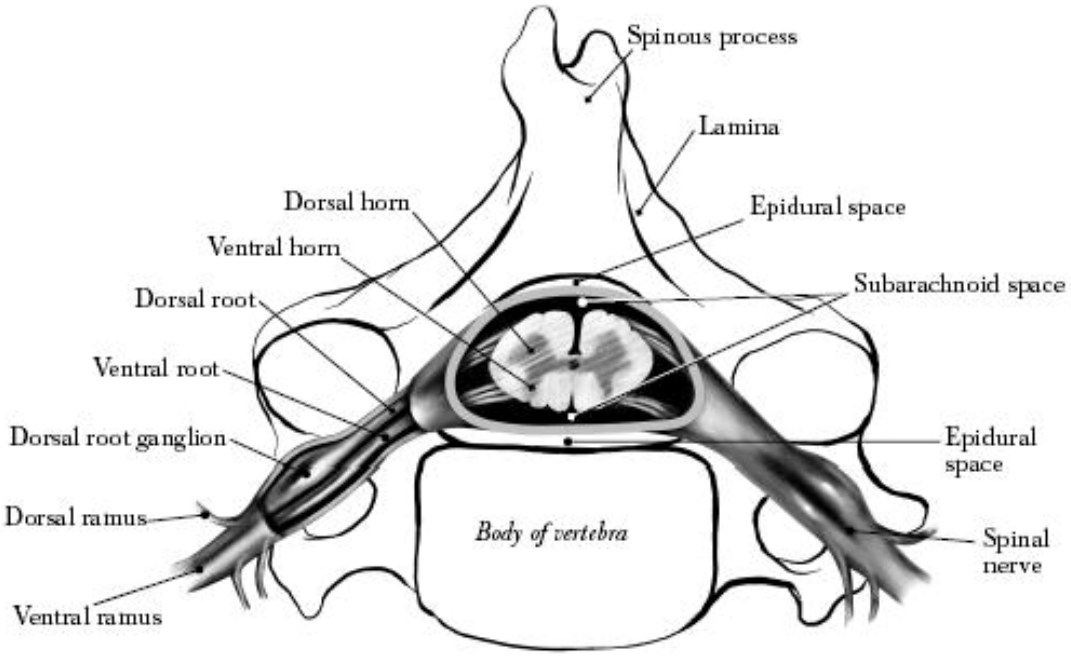
The spinal cord, a direct continuation of the medulla oblongata begins at the upper border of the atlas and terminates distally in the conus medullaris. The distal termination, because of the differential growth rates between the bony vertebral canal and central nervous

system varies from L3 in the infant, to the lower border of LI in the adult.

Surrounding the spinal cord in the bony vertebral column are three membranes (from within to the periphery), the piamater, arachnoid mater and duramater. The pia mater is a highly vascular membrane that closely invests the spinal cord. The arachnoid mater is a delicate neovascular membrane closely attached to the outermost duramater. Between the two innermost membranes is the subarachnoid space. In this space are the cerebrospinal fluid, spinal nerve roots, blood vessels that supply the spinal cord and the denticulate ligaments. Although the spinal cord ends at the lower border of LI in adults, the subarachnoid space continues to S2. The outermost membrane in the spinal canal is the longitudinally organized fibro elastic membrane, the duramater. This layer is the direct extension of the cranial duramater and extends as the spinal duramater from the foramen magnum to S2, where the filum terminale (an extension of the piamater beginning at the conus medullaris) blends with the periosteum of the subdural space which contains only small amounts of serous fluids to allow the dura and arachnoid move over each other.

Surrounding the duramater is the epidural space which extends from the foramen magnum to the sacral hiatus. Posterior to the epidural space is the ligamentum flavum which extends from the foramen magnum to the sacral hiatus. It runs from the anterior and inferior aspects of the lamina above to the posterior and superior aspect of the lamina below. Immediately posterior to the ligamentum flavum is the interspinous ligament. Extending from the external occipital protuberance to the coccyx, posterior to these structures is the supraspinous ligament. Lumbar puncture is routinely done below the L2 vertebrae down to the L5-S1 interspace to avoid damaging the spinal cord which ends at the lower border of LI in adults.


Epidural space (contains fat)
Subdural space
Subarachnoid space
Pia mater
Arachnoid
Dura mater (spinal dural sheath)
Spinal meninges
Spinothalamic tract conveying pain and temperature fibres


Spinous process
Lamina
Dorsal horn
Epidural space
Ventral horn
Subarachnoid space
Dorsal root
Ventral root
Dorsal root ganglion
Epidural space
Dorsal ramus
Body of vertebra
Spinal nerve
Ventral ramus

PHYSIOLOGY OF SUBARACHNOID BLOCK

The cerebrospinal fluid (CSF) is an ultrafiltrate of blood plasma with which it is in hydrostatic and osmotic equilibrium. It is a clear, colourless fluid found in the spinal and cranial subarachnoid space and in the ventricles of the brain. The average volume in the adult ranges from 120-150 ml of which 35 ml is in the ventricles, 25 ml is in the cerebral subarachnoid space and 75 ml is in the spinal subarachnoid space. It is secreted by the choroid plexus at a rate of 0.3-0.4 ml/minute.

Physical Characteristics of Cerebrospinal Fluid:

pH	**7.4**
Specific gravity (H_20) At body temperature At4°C	1.007 1.0003
Density	1.0003g/ml
Baricity	1.000
Pressure in supine position	8-12mm Hg
Cells	3-5/cu.mm
Proteins	20 mg/dl
Glucose	45-80 mg/dl

The cerebrospinal fluid plays an important role in spinal anesthesia as media for dispersion of the local anesthetic drug to the spinal nerve. An important factor determining the spread of drugs in the subarachnoid space is the specific gravity of the injected solution compared with that of CSF.

MECHANISM OF SPINAL ANAESTHESIA

Injection of local anaesthetic solution into the spinal CSF allows access to sites of action both within the spinal cord and the peripheral nerve roots. The nerve roots leaving the spinal canal are not covered by epithelium and are readily exposed to the local anaesthetic within the CSF. Therefore afferent impulses leaving via the ventral nerve roots are blocked during spinal anaesthesia. Local anaesthetics block sodium channels and propagation of action potential in spinal nerve roots. There are also multiple potential actions of local anaesthetics within the spinal cord at different sites. Local anaesthetics can exert sodium channel block with in the dorsal and ventral horns, inhibiting generation and propagation of electrical activity.

Zone of Differential Blockade:

Sensory:

In Subarachnoid block sympathetic fibers are blocked two to three segments higher than sensory fibers. Sympathetic block will be greater when more concentrated solutions are used or when adrenaline is added, as this has a similar effect.

Order of blocking nerve fibers:

1. Autonomic preganglionic B fibers.
2. Temperature fibers- Cold before warm.
3. Pinprick fibers.
4. Fibers conveying pain greater than pin prick.
5. Touch fibers.
6. Deep pressure fibers.
7. Somatic motor fibers.
8. Fibers conveying vibratory sense and proprioceptic impulses.

During recovery, sensations return in the reverse order, but it has been suggested that sympathetic activity returns before sensation.

SPREAD OF LOCAL ANAESTHETICS IN SUBARACHNOID SPACE

The local anaesthetic solution is diluted by CSF and therefore its original concentration is of less than the actual mass of drug injected. Spread is also determined by the baricity of the injected solution. Baricity is a ratio comparing the density of a local anaesthetic solution at a specified temperature to the density of CSF at the same temperature. A hypobaric solution has a baricity less than 1.0000 or specific gravity less than 1.0069 (the mean value of specific gravity). A hyperbaric solution has a baricity greater than 1.0000 or specific gravity more than 1.0069. Hypobaric and Hyperbaric solutions are prepared from isobaric solutions by the addition of various amounts of sterile distilled water and dextrose respectively. Isobaric solutions do not move under the influence of gravity in the CSF. Hyperbaric solutions, being heavier than CSF, settle to the most dependent aspect of the subarachnoid space, which is determined by the position of the patient. In supine patient,

hyperbaric solutions gravitate to the thoracic kyphosis. Hypobaric solution floats up against the gravity to the nerves innervating the surgical site.

FATE OF LOCAL ANAESTHETICS IN SUBARACHNOID SPACE

Following injection of local anaesthetic solution into subarachnoid space, its concentration falls rapidly. The initial steep fall is due to mixing with CSF and subsequent absorption into nerve roots and spinal cord. The egress of local anaesthetic solution following subarachnoid injection is primarily by vascular absorption. Depending on the type of the drug used, it is metabolized in plasma by pseudo cholinesterase or in the liver. As duration of anaesthesia is in part, a result of the rate of absorption from the subarachnoid space, the addition of a vasoconstrictor to the local anaesthetic solution will retard absorption of the drug and thus increase the duration of anesthesia.

INDICATIONS FOR SUBARACHNOID BLOCK

Spinal anaesthesia can be administered for surgeries below umbilicus.

- Lower abdominal surgeries,
- Lower limb surgeries,
- Urological procedures,
- Obstetric procedures,
- Gynecological surgeries,
- Perineal and rectal surgeries.

CONTRAINDICATIONS FOR SUBARACHNOID BLOCK

Absolute contraindication:

- Patient refusal.
- Local sepsis.

Relative contraindications:

- Coagulopathy.
- Fixed cardiac output states.
- Documented allergy to local anesthetics.
- Raised intracranial pressure.
- Neurological disease.
- Major spine deformities/previous surgery on the spine.
- Severe cardiac disease.

FACTORS INFLUENCING HEIGHT OF ANALGESIA IN SUBARACHNOID BLOCK

- Dose of the drug injected.
- Volume of fluid injected.
- Specific gravity of the solution.
- Position of the patient during injection.
- Posture of patient after injection.
- Choice of interspace.
- Patient factors- Age, Height, Pregnancy.

FACTORS NOT INFLUENCING HEIGHT OF ANALGESIA IN SUBARACHNOID BLOCK

- Patient factors- Weight, Sex.
- Barbatoge.
- Rate of injection.
- Composition and circulation of cerebrospinal fluid.
- Direction of bevel of the standard needle (although not of the Whitacare needle).

COMPLICATIONS OF SUBARACHNOID BLOCK

Immediate:

- Hypotension.
- Bradycardia.
- Toxicity due to intravascular injection.
- Allergic reaction to local Anesthetic.
- Hypoventilation (brain stem hypoxia).

Late:

- Post dural Puncture head ache.
- Retention of urine.
- Back ache.
- Meningitis.
- Transient lesions of cauda equina
- Sixth nerve palsy.
- Anterior spinal artery syndrome.
- Horner's syndrome.

CIRCULATORY EFFECTS OF SUBARACHNOID BLOCK

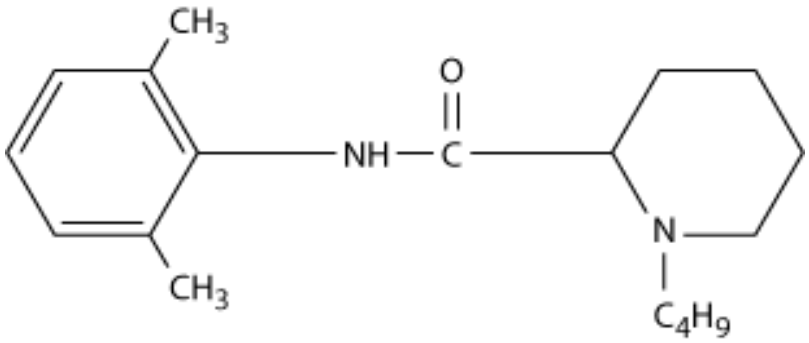
The subarachnoid block can influence the cardiovascular system, as follows.

1) Vasodilatation of resistance and capacitance vessels.
2) Block of cardiac efferent sympathetic fibers from T1 to T4 resulting in loss of chronotropic and inotropic drive and fall in cardiac output.

3) A further cause of bradycardia is the lowering of pressure in the right atrium consequent to diminished venous return (Bain Bridge reflex).
4) The operation of Marey's law causing tachycardia.
5) Vasodilatation and β-adrenergic blockade of myocardium with fall in cardiac output, following systemic absorption of the local anaesthetic drug.

PHARMACOLOGY OF BUPIVACAINE

Bupivacaine is an amide linked local anaesthetic. It is a hydrochloride of d(1)-1-butyl 2'6' pipecoloxylidide and is present as a racemic mixture.



It was synthesized by Eoaf Ekentem. First reports of its use was published in 1965 by Telinko. It's a very stable compound and may be autoclaved repeatedly. Its p_k is 8.1, molecular weight -288, protein binding 95%, lipid solubility 28%, elimination half life-210 minutes, toxic plasma concentration greater than 1.5 microgram/ml and approximate duration of action is 175 minutes.

AVAILABILITY

Ampoule-0.5% bupivaciane hydrochloride, 4cc with dextrose

-0.5% bupivacaine hydrochloride 20cc isobaric

Vials-0.25% and 0.5% bupivacaine hydrochloride 20cc

The maximum dose is 3mg/kg body weight

USES

Spinal anaesthesia

Epidural anaesthesia

Caudal anaesthesia

Continuous epidural anaesthesia

Peripheral nerve block

Local infiltration

PHARMACOKINETICS

Once injected intrathecally, it gets absorbed by the nerve rootlets and results in the desired effect. It is rapidly absorbed from the site of injection, but the rate of absorption depends on the vascularity at the site and presence of vasoconstriction. High lipid solubility of bupivacaine makes it easy for nerve and vascular tissue penetration.

80 to 85% of the absorbed bupivacaine gets into the plasma**.**

DISTRIBUTION

Rapid distribution phase(alpha)-in this phase the drug is distributed to highly vascular region half time being 2.7 minutes.

Slow disappearance phase(beta)-in this phase the drug distributes and slowly equlibriating tissues half time of Beta is 28 minutes.

Biotransformation and excretion phase- half time of γ is 3.5 hours.

BIOTRANSFORMATION

Possible pathways of metabolism of bupivacaine include aromatic hydroxylation and conjugation. Only the N-alkylated metabolite, N-butyl bupivacaine has been measured in blood or urine after epidural or spinal anaesthesia. Alpha acid glycoprotein is the most important plasma protein binding site of bupivacaine.

Excretion is through the kidney .4-10% of the drug is excreted unchanged.

MODE OF ACTION

a) Site of action-The spinal nerve rootlet fine nerve filaments having a large surface area are exposed to local anaesthetics.

b) Posterior and lateral aspects of the spinal cord

c) Sodium channel blockade-they impede sodium ion access to the axon interior by occluding the transmembrane sodium channels thus delaying the process of depolarization and axon remains polarized.

PHARMACODYNAMICS

CARDIOVASCULAR SYSTEM

Bupivacaine reduces cardiac output by reducing the sympathetic tone, by slowing the heart rate and by reducing the venous return, it produces a fall in arterial blood pressure but it is relatively slow and seldom it is very profound. It produces a fall in central venous pressure. It causes an increase in lower limb blood flow. It causes a reduction in incidence of deep vein thrombosis**.**

GASTROINTESTINAL TRACT

There is an increase in GIT motility.

TOXICITY

Toxicity is related to plasma level of unbound drug and more likely due to inadvertent intravenous injection. Systemic toxicity

reactions primarily involve CNS and CVS. The blood level required to produce CNS toxicity is less than that to produce circulatory collapse.

CENTRAL NERVOUS SYSTEM TOXICITY

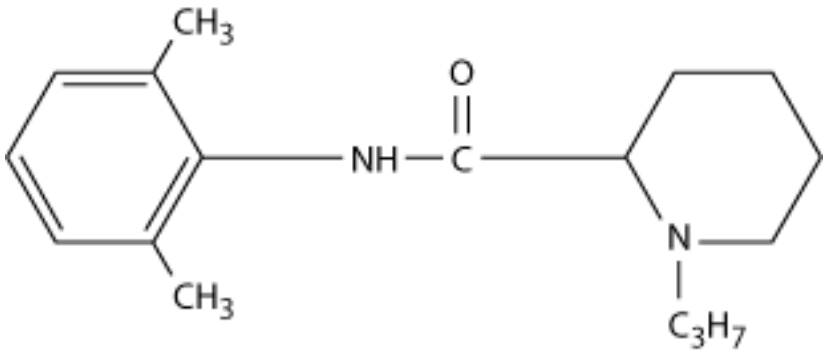
Initial symptoms include feeling of light headedness and dizziness, followed by visual and auditory disturbances. Objective signs are excitatory, muscle twitching and tremor, ultimately generalized tonic clonic seizure occurs**.**

CARDIOVASCULAR TOXICITY

The state of depolarization in fast conducting tissue of purkinge fibres of ventricular muscle is decreased. The rate of recovery of bupivacaine induced block is slower than that of lignocaine. Extremely high concentrations of the drug causes bradycardia and cardiac arrest.

PHARMACOLOGY OF ROPIVACAINE

In 1957, Ekenstam synthesized ropivacaine it's a new aminoamide local anaesthetic. It is s-1-propyl 2-6-pipecoloxylide hydrochloride mono hydrate, introduced clinically in 1996 has propyl group in piperdine nitrogen atom. It is long acting local anaesthetic, causes differential sensory and motor block. The rate depends on physiochemical properties-high pka and lipid solubility.pka is 8.1, lipid solubility and partial coefficient is 2.9.



Pka of bupivacaine and ropivacaine are identical, but ropivacaine is less fat soluble predicting that ropivacaine will block A fibres more slowly than bupivacaine.

It has chiral centre in piperidine ring with propyl group attached to nitrogen. It is single levo isomer s(-) enantiomer which has no asymmetric carbon. It is structurally related to bupivacaine and mepivacaine. It has dissociation constant of 8.1, protein binding of 90-

94%, lipid solubility is less than one half of bupivacaine, molecular weight of 328.89, weak vasoconstrictor.

MECHANISM OF ACTION

The clinical profile is similar to that of bupivacaine. It elicits nerve block via reversible inhibition of sodium ion influx in nerve fibres. This is potentiated by dose dependent inhibition of potassium channels. It blocks A delta and c fibres than motor fibres(being less lipophyllic).It has little motor block thus producing less incidence of deep thrombosis and better respiratory mechanics.

It is available in 0.2%, 0.5%, 0.75% and1% solution the maximum dose is 3mg per kg body weight and the toxic plasma concentration is 4 meq/L.

Dosage recommendations for ropivacaine in adults and children

Indication and procedure	Concentration (%)	Volume	Dose
In adults			
Surgical anaesthesia			
Lumbar epidural (caesarean section)	0.75	15-20 mL	113-150mg
Lumbar epidural (other surgery)	0.75	15-25mL	113-188mg
	1	15-20mL	150-200mg
Thoracic (single block for postoperative pain relief)	0.75	5-15mL	38-113mg
Intrathecal administration	0.5	3-4mL	15-20mg
Peripheral nerve block	0.75	10-40mL	75-300mg
Field block	0.75	1-30mL	7.5-225mg
Postoperative pain			

Lumbar epidural (continuous infusion)	0.2	6-10mL/h	12-20mg/h
Thoracic epidural (continuous infusion)	0.2	6-14mL/h	12-28mg/h
Peripheral nerve block (continuous infusion)	0.2	5-10mL/h	10-20mg/h
Filed block			
Intra-articular injection	0.2	1-100mL	2-200mg
Labour pain (lumbar epidural)	0.75	20mL	150mg
Bolus			
Intermittent top-ups	0.2	10-20mL	20-40mg
Continuous infusion	0.2	10-15mL	20-30mg
In children	0.2	6-14mL/h	12-28mg/h
Caudal epidural block (below T12)			
(For bodyweight up to 25kg)	0.2	1mL/kg	2mg/kg
Peripheral nerve block (e.g. ilioinguinal nerve block)	0.5	0.6mL/kg	3mg/kg

PHARMACOKINETICS-ABSORPTION AND DISTRIBUTION

The plasma concentration of ropivacaine depends on the total dose administered and the route of administration as well as the hemodynamic condition of the patient and the vascularity of the administered site. Ropivacaine is 94% bounded to alpha-1 acid glycoprotein. The duration of action depends on protein binding and clearance from the injection site. The volume of distribution of steady state is 59 litres; with a clearance ratio 0.73, hepatic extraction ratio of 0.4.The systemic toxicity is considered to be related to unbound drug concentration. The change in protein binding occurs with increase in plasma alpha acid glyco protein that accompanies the stress response to

surgery. Ropivacaine crosses the placenta but the foetal concentration is lower than maternal circulation.

OTHER EFFECTS

Ropivacaine inhibits platelet aggregation in plasma at concentration of 3.75 mg/ml. It has antibacterial activity in vitro inhibiting the growth of staphylococcus aureus, pseudomonas aeruginosa, E.coli.

ADVERSE EFFECTS

Hypotension, nausea, bradycardia, backpain, vomiting, headache, fever, chills, urinary retention, pruritus.

METABOLISM AND ELIMINATION

Ropivacaine is metabolised in liver by aromatic hydroxylation to3’-hydroxy-ropivacaine and N–deaikylation to 2’-6’-pipecoloxylidide. Other metabolites includes4’-hydroxy ropivacaine and 2’ hydroxy-methyl-ropivacaine. About 1-2% of drug is eliminated as unchanged in urine.

TOXICITY

Central nervous system toxicity is directly related to local anaesthetic potency and the convulsant doses of ropivacaine and bupivacaine are similar. Local anaesthetics exert their direct toxic effects on heart by blocking sodium influx through sodium channels. This causes depression of maximal rate of increase (Vmax) of cardiac action potential and results in delayed conduction, ropivacaine depress Vmax less than bupivacaine and recovery is quicker after ropivacaine.

MATERIAL AND METHODS

After approval of the study by the Institutional Ethical Committee the study was conducted in , 100 ASA physical status I-II patients, undergoing elective lower abdominal surgery under spinal anesthesia. The age of the patients ranged from 23-68 yrs weighing 35-65kgs and height ranging from 150-168 cms. All patients were thoroughly examined preoperatively. Informed written consent was obtained and the procedure was explained. All patients weight, height were noted.

In the assessment room, vital parameters like pulse rate, blood pressure and baseline investigations like Haemoglobin, urine analysis for albumin, sugar ,blood sugar urea, creatinine and ECG were checked. Thorough examination of all the system and airway assessment was done.

Exclusion criteria included, local infection, bleeding disorder. The patients were randomly allocated into two groups of 50 each.

Group A patients received 3ml of 0.5% isobaric ropivacaine (15mg).

Group B patients received 3ml of .5% isobaric bupivacaine (15mg) 5mg/ml.

The total volume of the injected solution was 3ml in both groups. In the operating room, appropriate equipment for airway management and emergency drugs were kept ready. Patients were shifted to the operating room. The horizontal position of the operating table was checked. NIBP monitor, Pulseoximetry and ECG leads were connected to the patient. Preoperative baseline mean arterial pressure, pulse rate and O2 saturation were recorded. Patients were cannulated with 18G intravenous cannula and preloaded with 1000 ml of ringer lactate. The patients were placed in right lateral position. The skin over the back was prepared with antiseptic solution and draped with sterile towel. Lumbar puncture was performed with a25G spinal needle at L2-L3 or L3-L4 interspace via midline approach. After confirming free flow of CSF, the drug was injected. The patients were made to lie supine immediately after injection and the time at which the drug injected was noted.

THE FOLLOWING PARAMETERS WERE OBSERVED

Sensory block

The onset of sensory block was defined as the time between the injection of anaesthetic solution and the absence of pain to pinprick at the T10 dermatome. Sensory block was assessed by loss of sensation. (pin prick sensation using 21G sterile needles) bilaterally along the midclavicular line. This assessment started immediately after turning the patient supine and continued every minute till the peak block height was reached and the time was noted. The duration of sensory block was defined as the time for regression of two segments from the maximum level of sensory block height evaluated by the pinprick. Sensory block was checked every 15 mts till it reached two segment regression.

MOTOR BLOCK

Motor block was assessed bilaterally using Modified Bromage scale.

MODIFIED BROMAGE SCALE.

0-no block, able to raise extended legs against gravity.

1-unable to raise extended leg, but just able to flex knees.

2-unable to flex knees, but able to flex ankles

3-Total block-inability to flex ankle

Assessment of motor block was started immediately after turning the patient supine and continued every minute till Bromage score of 3 was reached. The onset of motor block was defined as the time to achieve Bromage score of 3 from the time of injection. Duration for complete motor block recovery was taken as the time from subarachinoid injection to return of Bromage score to zero.

VITAL SIGNS AND SIDE EFFECTS

Mean arterial pressure, pulse rate were recorded every two minutes for the first 10 mts and thereafter every 5 mts until the immediate post operative period Oxygen saturation monitored continuously. Hypotension was defined as a fall in systolic blood pressure more than 30% from baseline or systolic blood pressure less than 90mm Hg. This was managed by incremental doses of 6mg intravenous ephedrine.

Bradycardia was defined as heart rate less than 60/mt and managed by incremental doses of 0.3mg intravenous atropine. Respiratory depression was said to be present if respiratory rate was less than 8 /mt and $SP0_2$ less than 85%. The respiratory arrest was managed

with intubation and IPPV. Vomiting was managed with metaclopramide 10 mg intravenously. Pruritis was managed with reassurance or by pheniramine maleate 22.75 mg. Urinary retention was monitored postoperatively and catheterization was planned in patients with prolonged retention more than 6 hours. Patients were shifted to recovery room after completion of surgery.

STATISTICAL TOOLS

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using **Epidemiological Information Package.**

Using this software range, frequencies, percentages, means, standard deviations, chi square and 'p' values were calculated. Kruskul Wallis chi-square test was used to test the significance of difference between quantitative variables. The 'p' value less than 0.05 is taken to denote significant relationship.

REVIEW OF LITERATURE

P.D.W.Fettes, G.Hocking et. al., conducted double blinded prospective randomised study in 40 patients undergoing elective perineal surgery under spinal anaethesia. They received either 15mg of plain ropivacaine or 15mg of hyperbaric ropivacaine. He reported that intrathecal plain ropivacaine produced satisfactory analgesia. The study showed the cephalic spread to T8 in plain and T4 in hyperbaric group respectively. There was a significant difference in the onset of sensory block to T10 of about 10 minutes for plain and 5 minutes for hyperbaric groups respectively. The motor regression time are 180 minutes for plain and 120 minutes for hyperbaric groups respectively, showing hyperbaric ropivacaine produced more rapid onset of motor block which ultimately regress more quickly.

M. Mantouvalou S Rali et al in their study compared the anaesthetic efficacy and safety of 3 local anaesthetic agents namely 15mg of racemic bupivacaine, ropivacaine and levobupivacaine in patients undergoing lower abdominal surgeries. It stated that cephalic spread of sensory block was similar in all groups with onset of motor

block in bupivacaine is 8 ± 5 minutes and ropivacaine 12 ± 5 minutes. Thus it showed faster onset with bupivacaine and shorter duration of motor block in ropivacaine of 100 ± 34 minutes when compared to bupivacaine of 150 ± 40 minutes.

H.Kallio, E,V.T Snall et. al., in the prospective randomized double blinded study in 90 ambulatory lower extremity surgery patients who received 2 ml of isobaric ropivacaine 1%, 0.75% and isobaric 0.5% bupivacaine. It showed adequate block level with hemodynamic stability and faster motor recovery of about 137.2 minutes in ropivacaine when compared to bupivacaine (204.4 minutes).

Jean-Marc., Malinovsky, Florence Charles, Ottman Kiel et al compared 15mg of intrathecal isobaric ropivacaine with 10mg isobaric bupivacaine in patients scheduled for transurethral resection of bladder or prostate. The cephalic spread in bupivacaine is T7 and ropivacaine is T9. The onset of motor block of ropivacaine is 11 minutes and its recovery is 105 ± 25 minutes where as in bupivacaine 13 ± 8 minutes and 127 ± 17 minutes respectively. No difference in hemodynamic effects was detected between the two groups.

Kim, S. Khaw, Warwick d.ngen kee et al conducted a comparative study of 25 mg spinal hyperbaric and isobaric solution of ropivacaine in caesarean sections. It showed more extensive spread (T4), faster onset of motor block (7.7 minutes) and its faster recovery (144 minutes) in hyperbaric group than isobaric ropivacaine (T7 and 13.8 minutes, 184 minutes)and reported the onset of sensory block of 11.4 mins and its duration 216 mins .

D.A. McNamee, A.M.Mc Clellana, S.Scot et. al., in their comparative study of the efficacy and safety of 17.5mg of plain ropivacaine with 17.5mg of plain bupivacaine for spinal anaesthesia in patients undergoing total hip arthroplasty stated that there was a trend for patients in bupivacaine to achieve higher upper dermatomal level of sensory block but this difference was not significant. There was no significant difference in the median time of onset of sensory block at T10 dermotome which was 2 minutes with ropivacaine and bupivacaine. Duration of sensory block longer in bupivacaine (3.5 hours) compared to ropivacaine (3 hours). There is a shorter duration of motor block in ropivacaine 2.1 hours versus 3.9 hours in bupivacaine.

P. Gautier M. Dekock L. Huberty T Demir compared the effects of intrathecal isobaric ropivacaine, levobupivacaine and bupivacaine for caesarean section in 90 parturients. Combined spinalepidural technique was used with bupivacaine (8mg), leveobupivacaine (8mg), ropivacaine (12mg) all combined with sufentanil 2.5microgram. In the study, the onset and duration of motor block in bupivacaine was 9 and 142 minutes and with ropivacaine 14 and 116 minutes, showing shorter duration of motor block with ropivacaine. The duration of sensory block with bupivacaine was 145 minutes and ropivacaine was 135 minutes.

OBSERVATION AND RESULTS

All 100 patients in two groups completed the study without any exclusion. We did an inter group analysis and the results were as followed. Of the 100 patients 50 belonged to Group A (ropivacaine) and other 50 categorized as Group B(bupivacaine). Data were presented as range, mean, standard deviation. The probability value ‘P’ of less than 0.05 considered statistically significant.

Table 1 : AGE DISTRIBUTION

Age group	**Group A**		**Group B**	
	No.	**%**	**No.**	**%**
Less than 20 yrs	2	4	1	2
20-29 yrs	11	22	5	10
30 – 39 yrs	12	24	11	22
40 – 49 yrs	14	28	14	28
50-59 years	6	12	11	22
60 years & above	5	10	8	16
Total	50	100	50	100
Range Mean S.D. Value	19 – 66 years 40.0 years 13 0 years 40 ±13		19 – 68 years 45 years 12.5 years 45 ±12.5	
'p'	0.0543 Not significant			

Patients demographic data between the two group were comparable. In table1 illustrated the age distribution in the range of 19-66 years in Group A and 19-68 years in Group B. The mean, age was statiscally similar. 'p' value is 0.543. which was statistically insignificant.

Table 2 : SEX DISTRIBUTION

Sex	Group A		Group B	
	No.	**%**	**No.**	**%**
Male	35	70	36	72
Female	15	30	14	28
Total	50	100	50	100
'p'	0.8264 Not significant			

Table 2 represents sex distribution in our study. Of the 100 patients 71 were male and 29 were female. Group A has 70% male and 30% female. Group B has 72% male and 28% female. The distribution was similar in both groups. The p value is 0.8264 which was insignificant.

TABLE 3 :

DURATION OF SURGERY (IN MINUTES)

Duration of Surgery (in minutes)	**Group A**	**Group B**
Range	45– 160 minutes	45 – 125 minutes
Mean	94 minutes	97.4 minutes
S.D.	22.2 minutes	12.9 minutes
Value	94 ± 22.2	97.4 ± 12.9
'p'	0.4924 Not Significant	

The average duration of surgery in both groups were comparable. The 'p' value of 0.4924 which was not significant.

Table 4 : PEAK SENSORY LEVEL

Peak Sensory Level	Group A		Group B	
	No.	**%**	**No.**	**%**
T2	-	-	-	-
T4	1	2	-	-
T6	1	2	16	32
T8	45	90	34	68
T12	3	6	-	-
Total	50	100	50	100

In this table the distribution of upper extend of sensory block in both groups were given. T10, the ideal peak sensory level is attained.

Table 5: GRADING OF MOTOR BLOCK

Grading of Motor Block	Group A		Group B	
	No.	%	No.	%
0	-	-	-	-
1	-	-	-	-
2	22	44	-	-
3	28	56	50	100
Total	50	100	50	100
'p'	**0.0001 Significant**			

In group A 44% attained grade 2 block and 56% attained grade 3 block.

In group B 100% attained grade 3 block.

Table 6 : TIME OF ONSET OF SENSORY BLOCK

(IN MINUTES)

Time of onset of sensory block **(in minutes)**	**Group A**	**Group B**
Range	5 – 15 minutes	3 – 7 minutes
Mean	10.2 minutes	4.2 minutes
S.D.	2.8 minutes	1.0 minutes
Value	10.2 ± 2.8	4.2 ± 1.0
'p'	**0.0001** **Significant**	

The average time taken for onset of sensory block is 10.2 minutes in group A and 4.2 minutes was shown in group B. The p value was 0.0001 which was significant.

Table 7 : TOTAL DURATION OF SENSORY BLOCK

(IN MINUTES)

Total duration of Sensory Block (in minutes)	**Group A**	**Group B**
Range	100– 230 minutes	135 – 180 minutes
Mean	145.9 minutes	152.8 minutes
S.D.	34.8 minutes	9.1 minutes
Value	145.9 ± 34.8	152.8 ± 9.1
'p'	0.145 Not Significant	

In this table the duration of sensory block in group A was 145.9 minutes and in group B is 152.8 minutes. The p value was 0145 which was not significant.

Table 8 : ONSET TIME OF MOTOR BLOCK (IN MINUTES)

Onset Time of Motor Block (in minutes)	**Group A**	**Group B**
Range	10– 20 minutes	6 – 10 minutes
Mean	14.3 minutes	9.3 minutes
S.D.	3.1 minutes	1.0 minutes
Value	14.3 ± 3.1	9.3 ± 1.0
'p'	**0.0001 Significant**	

In Table 8 and 9 the onset of motor block distribution in both groups were depicted. The p value was 0.0001, which was significant. The onset of motor block (time to achieve a bromage score of 3) was significantly faster in bupivacaine group of 9.3 minutes when compared with ropivacaine of 14.3 minutes.

Table 9 : TOTAL DURATION OF MOTOR BLOCK (IN MINUTES)

Total duration of Motor Block (in minutes)	**Group A**	**Group B**
Range	80– 240 minutes	100 – 300 minutes
Mean	137.2 minutes	204.4 minutes
S.D.	35.5 minutes	37.2 minutes
Value	137.2 ± 35.5	204.4 ± 37.2
'p'	**0.0001 Significant**	

The table shows shorter duration of motor block in group A of 137.2 ± 35.5 with significant p value.

Table 10 : BASELINE HEMODYNAMICS

Parameter	Group A		Group B		'p'
	Mean	**S.D.**	**Mean**	**S.D.**	
Pulse rate	85.7	17.0	84.3	14.6	0.7587 Not significant
Respiratory rate	13.36	1.68	13.16	1.3	0.6141 Not significant
Mean Arterial Pressure	87.3	17.5	91.3	18.1	0.2323 Not significant

The study showed stable hemodynamic status with decreased incidents of hypotension and insignificant 'P' value.

Table 11 : SPO_2

SPO2	Group A	Group B
Range	97 – 99	97 – 99
Mean	97.98	98.04
S.D.	0.59	0.45
Value	97.98 ± 0.59	98.04 ± 0.45
'p'	0.8009 Not Significant	

DISCUSSION

M. Mantouvalou S Rali et al in the study to compare the aneasthetic efficacy and safety of three local anaesthetic agents namely 15mg of racemic bupivacaine, ropivacaine and levobupivacaine in patients undergoing lower abdominal surgeries showed no significant difference in duration of sensory block between the groups. It stated delayed onset of 12 ± 5 minutes and faster recovery of motor block of 100 ± 34 minutes in ropivacaine group as seen in our study.

Kim S.Khaw et al., in their study compared 25mg of intrathecal hyperbaric and isobaric solution of ropivacaine in caesarean section. The onset of sensory block in isobaric group was 11.4 mins which was comparable to our study of 10 mins. Similarly the onset of motor block in isobaric ropivacaine group was 13.8 mins which was comparable to our study of 14.3 mins. The duration of sensory block and motor block was 216 mins and 184 mins where as in our study it was 145.2 mins and 137.2 mins and the difference in this results was due to usage of higher dose of 25mg of isobaric ropivacaine in their study.

Jean Marc, Malinovsky et al in their study compared 15mg of intrathecal isobaric ropivacaine with 10mg of isobaric bupivacaine in patients scheduled for transurethral resection of bladder or prostate. The study showed no difference in hemodynamic effects between their groups as correlated with our study. The study reported similar intensity and the duration of motor block with isobaric ropivacaine was 165 mins and that of bupivacaine was 184 mins. The difference in the duration in the motor block in the above study is comparable to our results.

Helena Kallio et al., conducted a study in 90 ambulatory lower extremity surgery patients who received plain solution of 2ml of 1% , 0.75% ropivacaine and 0.5% bupivacaine. It reported the median onset and duration of motor block in 15mg plain ropivacaine group was 20 mins and 150 mins which were comparable to 14.3 mins and 137.2 mins in our study. The onset and duration of sensory block in 15mg ropivacaine group was 10 mins and 150 mins which were comparable to our study of 10 mins and 145.2 mins. The rationale for dose selection in this study was due to duration of action of ropivacaine in spinal anesthesia is approximately 50% to 67% that of bupivacaine, so that

patients could mobilise faster in both ropivacaine and bupivacaine groups.

Furthermore, Gautier et al compared the effects of intrathecal administration of 8mg of isobaric bupivacaine, 8mg levobupivacaine or 12mg ropivacaine all combined with sufentanil 2.5micrograms in patients undergoing caesarean delivery. Gautier selected the dose in his study under the hypothesis that three times of ropivacaine dose is equivalent to two times of bupivacaine. The onset and duration of motor block in ropivacaine group of their study was 14 mins and 116 mins which was comparable to 14.3 mins and 137.2 mins in our study. The onset and duration of motor block in bupivacaine group of their study was 9 mins and 142 mins where as it was 4 mins and 204 mins in our study. The difference in the duration of motor block between their and our study may be due to lower dose of bupivacaine used in their study.

P.D.W fettes, G. Hocking et al conducted a study in 40 patients undergoing elective perineal surgery under spinal anaesthesia receiving either 15mg of plain ropivacaine or 15mg of hyperbaric ropivacaine. The onset of sensory block in isobaric ropivacaine group was 10 mins

which was comparable to our study of 10 mins. The duration of motor and sensory block in isobaric ropivacaine group was 180 mins and 270 mins. Thus showing shorter duration of motor block than sensory block in ropivacaine as in our study. The study showed rapid onset of motor block of 5 mins and its recovery of 120 mins. The difference in the speedy onset and recovery of motor block in hyperbaric versus isobaric ropivacaine group is attributed to the baricity of the solution.

McNamee et al compared 17.5mg of plain ropivacaine with 17.5 mg plain bupivacaine in patients undergoing total hip arthroplasty under spinal anaesthesia. The onset of sensory block in both groups was 2 mins. But in our study, it was 10 mins in ropivacaine and 4 mins bupivacaine. The difference in due to method of testing of sensory loss to ice in the above study. The duration of motor block is 130 mins in ropivacaine group which was comparable to our study of 137.2 mins. The duration of motor block in bupivacaine group was 230 mins where as 204mins in our study. Thus the above study showing shorter duration motor block in ropivacaine group as seen in our study. The duration of sensory block in the study was 180 mins in ropivacaine group and 110 mins in bupivacaine group where as 145.9 mins and 152 mins

respectively in our study. The difference in the duration of motor and sensory block of bupivacaine group between their and our study may be attributed to different dosage used in their study.

SUMMARY

This study was conducted to compare the anaesthetic efficacy of intrathecal isobaric 0.5% ropivacaine with isobaric 0.5% bupivacaine in lower abdominal surgeries, in 100 ASA grade I and II patients of both sexes in the age group of 19-68 years undergoing elective lower abdominal surgery under spinal anaesthesia. After getting ethical committee approval and informed written consent from the patients, 100 patients were allocated into two groups of 50 patients each. The baseline pulse rate and mean arterial pressure were recorded. The first group A received 3ml of isobaric 0.5% ropivacaine (5mg/ml) and the second group received 3ml of isobaric 0.5% bupivacaine intrathecally. The pulse rate, mean arterial pressure , the onset of sensory and motor block, duration of sensory and motor block were recorded in both groups. The results were analysed statistically using epidemiologically information package.

On comparison of data we have found that the intrathecal isobaric 0.5% ropivacaine produces delayed onset, but similar duration of sensory block and a statistically significant shorter duration of motor block. The hemodynamics and height of block similar in both groups.

CONCLUSION

We conclude from this study, that the intrathecal administration of 3ml of 0.5% isobaric ropivacaine when compared to 3ml of 0.5% isobaric bupivacaine, produces delayed onset but similar duration of sensory block (analgesia), and a statistically significant shorter duration of motor block. The hemodynamics and height of block (peak sensory level) are similar.

Though the onset of sensory block is delayed, because of the effects on motor block, we consider ropivacaine can be a better choice for ambulatory anaesthesia.

PROFORMA

NAME: Age /Sex: I.P.No: Ht: Wt:

Duty Anaesthesiologist:

ASA Physical Status:

PR: BP: RR: CVS:

RS:

Blood Investigations:

Hb%: RBS: Urea: creatinine grouping & Typing

Intraop - Group A & B

Position and site of Injection :

Time of Injection of drug :

Volume of drug injected :

Time of onset of sensory block- Loss of sensation with pinprick test with 21 G im needle.

Maximum level of cephalad block:

Time of onset of motor block :

Duration of Sensory block :

Duration of procedure :

Degree and duration of motor block:

Assessed by Bromage scale :

‘0’ - No motor block (Movement of limbs) full flexion of knees, feet able to lift extended leg.

‘1’ - Unable to lift the extended leg. (Just flex knees and feet) not hip.

‘2’ - In ability to flex knees, but flexion of feet possible.

‘3’ - In ability to flex ankle,unable to move leg, complete motor paralysis.

PR: Mean Arterial Pressure: RR: SPO2:

Side effects and Complication:

Insufficient block :

Any discomfort : Nausea, vomiting,pruritis,pain

Hypo tension > 30% fall systoloc Bp

Bradycardia < 50/mt

Shivering

Post of Headache & Backache

MASTER CHART

No	GROUP	Age	Sex	IP No	DM	HTN	IHD	COPD	OTHERS	PR	RR	MAP	Time of onset of S.B.	Peak sensory leve l	Total duration of S.B.	Time of onset of MB	Total duration of MB	grade of motor block	Duration of surgery	SPO2	ADVERSE EFFECTS
1	A	22	F	1092597	-	-	-	-	-	88	14	108	5.5	T8	200	10	240	3	95	98	
2	A	25	M	1094118	-	-	-	-	-	81	12	108	7	T8	145	12	150	3	100	98	
3	A	35	F	1095381	-	-	-	-	-	90	16	90	6.5	T8	110	16	180	3	120	98	
4	A	28	F	1095403	-	-	-	-	-	108	12	60	10	T6	100	13	200	3	90	98	Hypotension
5	A	19	M	1095388	-	-	-	-	-	72	13	70	12	T12	150	15	90	2	110	97	Insufficient Block
6	A	29	M	1092590	-	-	-	-	-	86	15	74	11	T8	170	18	130	3	120	98	
7	A	45	F	1094110	-	-	-	-	-	70	14	78	6	T8	130	10	180	3	95	98	
8	A	60	M	1095413	1	-	1	-	-	70	12	90	13	T8	115	12	170	3	100	98	
9	A	40	M	1095340	-	-	-	-	-	68	12	103	10	T8	200	15	160	3	90	98	
10	A	39	M	1094000	-	-	-	-	-	90	11	108	7	T8	180	14	120	3	120	99	
11	A	20	F	1092595	-	-	-	-	-	100	12	58	9	T8	100	11	100	3	110	98	Hypotension
12	A	32	M	1094114	-	-	-	-	-	78	14	110	12	T8	110	10	120	3	90	98	
13	A	19	M	1092598	-	-	-	-	1	88	16	96	14	T8	140	10	96	3	100	98	
14	A	66	M	1094116	-	1	-	1	-	118	16	56	5	T8	115	12	135	3	120	98	
15	A	33	F	1090952	-	-	-	-	-	86	12	92	8	T8	120	12	100	3	95	98	Hypotension
16	A	28	M	1092500	-	-	-	-	-	90	14	112	15	T8	180	15	150	3	100	98	
17	A	45	M	1094008	-	-	-	1	-	74	12	110	13	T8	140	20	180	3	110	99	
18	A	42	M	1095400	-	-	-	-	-	70	13	88	11	T8	120	16	135	3	120	98	
19	A	59	M	1094111	1	-	-	-	-	68	16	80	10	T12	160	18	100	2	90	98	Insufficient Block
20	A	38	F	1095421	-	-	-	-	-	64	15	70	12	T8	160	10	140	3	95	98	
21	A	45	M	1095499	-	-	-	-	-	84	14	92	10	T8	115	10	140	3	95	98	
22	A	40	M	1095361	-	-	-	-	-	90	12	90	9	T8	180	12	160	3	95	98	
23	A	23	M	1094021	-	-	-	-	-	118	11	60	7	T2	180	16	110	3	100	99	
24	A	36	M	1092596	-	-	-	-	-	130	13	61	6	T8	160	15	110	3	120	98	Hypotension
25	A	45	F	1095431	-	-	-	-	-	94	15	103	13	T8	150	18	180	3	140	98	Hypotension
26	A	47	F	1095371	1	-	-	-	-	88	14	105	15	T8	100	20	160	3	100	98	
27	A	65	M	1092561	-	1	-	-	-	66	16	70	14	T8	200	16	98	3	90	98	
28	A	35	M	1095499	-	-	-	-	-	116	10	80	9	T8	120	20	110	3	160	97	

29	A	30	M	1095380	-	-	-	-	-	90	12	84	13	T8	140	10	140	3	60	98	
30	A	51	M	1094331	-	1	-	1	-	100	13	110	12	T8	200	14	115	3	70	98	
31	A	36	M	1095331	-	-	-	-	-	84	16	102	11	T12	100	15	160	2	85	98	
32	A	28	F	1095402	-	-	-	-	-	90	14	108	9	T8	120	12	80	2	90	98	Insufficient Block
33	A	46	M	1094008	-	-	-	-	-	72	12	98	10	T8	120	14	120	2	115	99	
34	A	60	M	1092590	-	-	1	-	-	70	11	86	10	T8	100	18	110	2	60	98	
35	A	57	M	1095300	-	1	-	1	-	88	13	74	15	T8	150	18	90	2	80	98	
36	A	40	F	1095381	-	-	-	-	-	118	14	62	9	T8	230	14	160	2	45	98	
37	A	38	M	1095431	-	-	-	-	-	74	16	78	12	T8	100	12	190	2	60	97	Hypotension
38	A	22	F	1094312	-	-	-	-	-	120	15	57	6	T8	160	18	185	2	65	98	
39	A	43	M	1092583	-	-	-	-	-	116	14	108	8	T8	200	16	175	2	75	98	Hypotension
40	A	34	M	1094009	-	-	-	-	-	68	12	102	10	T8	170	12	105	2	80	98	
41	A	59	M	1095370	-	-	-	-	-	70	11	110	9	T8	140	13	100	2	100	98	
42	A	46	F	1095414	-	-	-	-	-	84	12	90	11	T8	120	15	154	2	65	99	
43	A	28	M	1095300	-	-	-	-	-	80	12	68	13	T8	115	18	130	2	60	98	
44	A	36	M	1095418	1	-	-	-	-	78	16	70	10	T8	140	11	140	2	60	98	
45	A	60	F	1095384	-	-	-	1	-	72	14	100	15	T8	120	15	160	2	100	98	
46	A	58	M	1095411	-	-	-	-	-	70	11	102	13	T8	190	20	130	2	110	98	
47	A	43	F	1093396	-	-	-	-	-	86	12	70	9	T8	180	11	100	2	75	98	
48	A	27	M	1095312	-	-	-	-	-	74	13	80	10	T8	170	12	100	2	85	97	
49	A	59	M	1095391	-	-	-	-	-	68	14	84	6	T8	100	15	180	2	90	98	
50	A	40	M	1094021	-	-	-	-	-	70	15	100	10	T8	180	18	95	2	100	98	
51	B	30	F	1092241	-	-	-	-	-	88	12	109	4	T8	155	8	100	3	85	98	
52	B	32	M	1094401	-	-	-	-	-	72	14	60	3	T6	150	9	185	3	120	98	
53	B	22	M	1095503	-	-	-	-	-	90	16	78	5	T6	155	10	225	3	110	98	
54	B	25	F	1096402	-	-	-	-	-	98	14	70	6	T8	150	8	165	3	115	97	Hypotension
55	B	27	M	1092201	-	-	-	-	-	78	11	80	3	T8	155	9	240	3	100	98	
56	B	30	M	1093567	-	-	-	-	-	60	13	90	4	T8	150	10	190	3	90	98	
57	B	36	M	1092234	-	1	-	1	-	58	14	92	3	T6	145	10	210	3	95	98	Shivering
58	B	42	F	1093421	-	-	-	-	-	101	15	110	5	T6	135	6	260	3	105	98	
59	B	19	F	1094562	-	-	-	-	-	56	16	106	6	T6	165	10	240	3	110	99	

60	B	56	M	10933227	-	-	-	-	-	60	12	105	6	T8	155	10	230	3	120	98	Shivering
61	B	29	F	1092634	-	-	-	-	-	62	12	60	5	T8	145	10	180	3	95	98	
62	B	63	M	1092464	1	-	-	-	-	79	14	106	3	T6	165	8	160	3	100	98	
63	B	46	M	1093404	-	-	-	-	-	74	13	100	4	T8	160	10	150	3	115	98	
64	B	40	F	1095545	-	-	-	-	-	96	11	103	5	T8	150	10	160	3	125	98	Hypotension
65	B	54	M	1092586	-	-	-	-	-	80	12	104	3	T8	140	10	180	3	120	98	
66	B	55	M	1092602	-	-	-	-	-	84	13	110	4	T8	155	10	190	3	105	99	Shivering
67	B	58	M	1093495	-	-	-	-	-	76	14	60	5	T8	150	8	195	3	85	98	
68	B	60	M	1092293	1	1	-	1	-	60	14	106	3	T8	180	10	210	3	90	98	Shivering
69	B	29	F	1093413	-	-	-	-	-	110	14	108	3	T6	165	10	160	3	75	98	
70	B	36	M	1092753	-	-	-	-	-	78	12	106	3	T8	160	10	190	3	90	98	
71	B	41	F	1094424	-	-	-	-	-	62	11	104	4	T8	155	8	210	3	105	98	Hypotension
72	B	37	M	1095453	-	1	-	-	-	82	14	64	5	T6	160	9	215	3	110	97	
73	B	42	M	1092376	-	-	-	-	-	88	12	90	6	T8	150	10	220	3	120	98	
74	B	53	M	1093534	-	-	-	-	-	90	13	78	7	T8	170	10	240	3	95	98	
75	B	61	M	1094615	-	-	-	-	1	92	12	84	3	T8	165	10	225	3	105	98	Hypotension
76	B	55	M	1092661	-	-	-	-	-	78	14	64	3	T8	155	10	225	3	90	99	
77	B	68	M	1093552	-	-	-	-	-	99	15	90	4	T6	160	8	200	3	95	98	
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86	B	40	F	1094603	1	1	-	-	-	96	13	110	5	T8	150	10	180	3	115	98	
87	B	60	M	1092544	-	-	-	-	-	76	11	60	5	T8	155	10	185	3	85	98	
88	B	49	M	1092496	-	-	-	-	-	108	14	78	3	T8	145	10	200	3	80	98	Hypotension
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90	B	64	M	1094377	-	-	-	-	-	88	14	96	5	T6	145	9	180	3	85	99	
91	B	40	F	1095574	-	1	-	-	-	90	14	84	4	T6	165	10	250	3	100	98	

92	B	61	M	1094406	-	-	-	-	-	120	14	80	5	T6	155	9	265	3	80	98	
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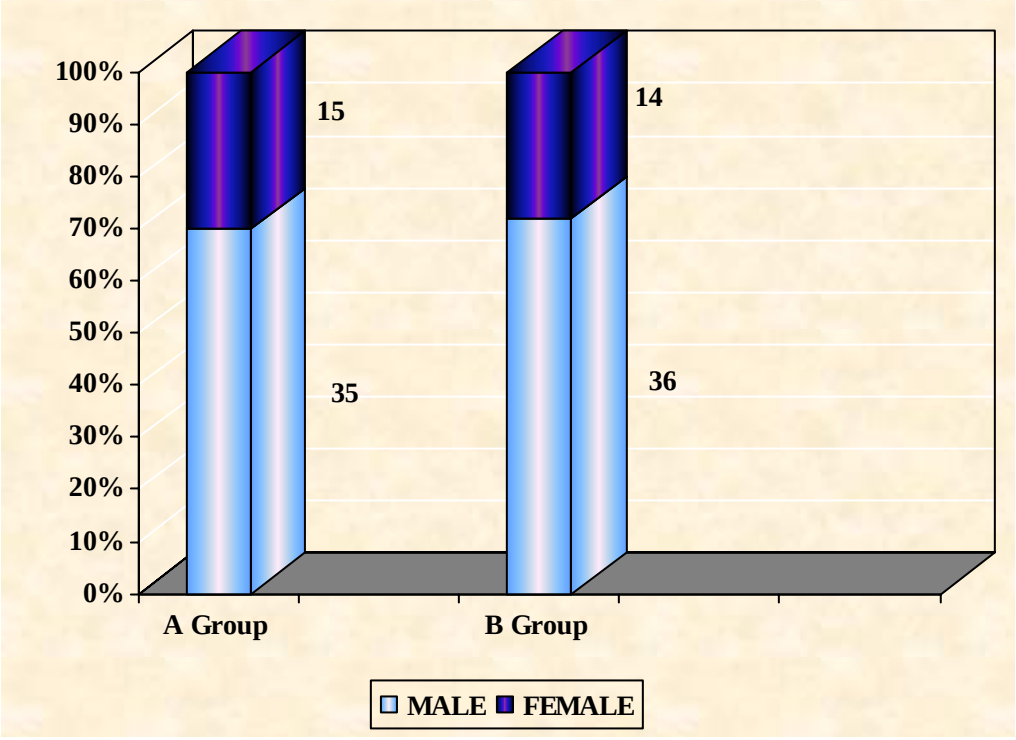

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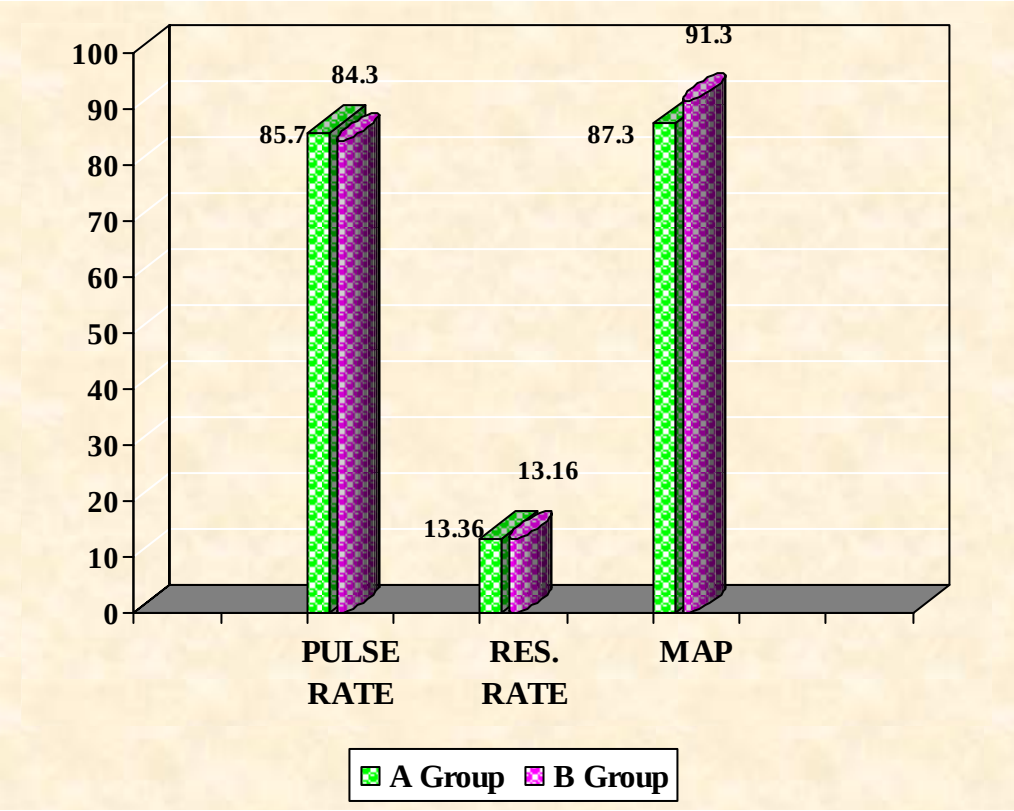

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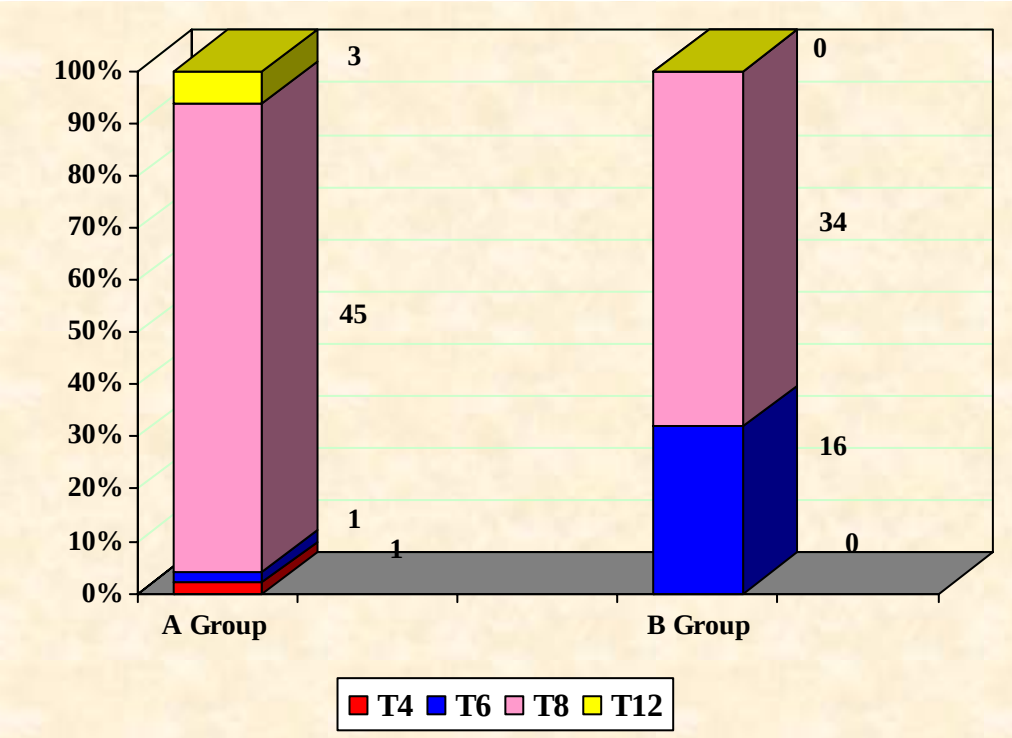

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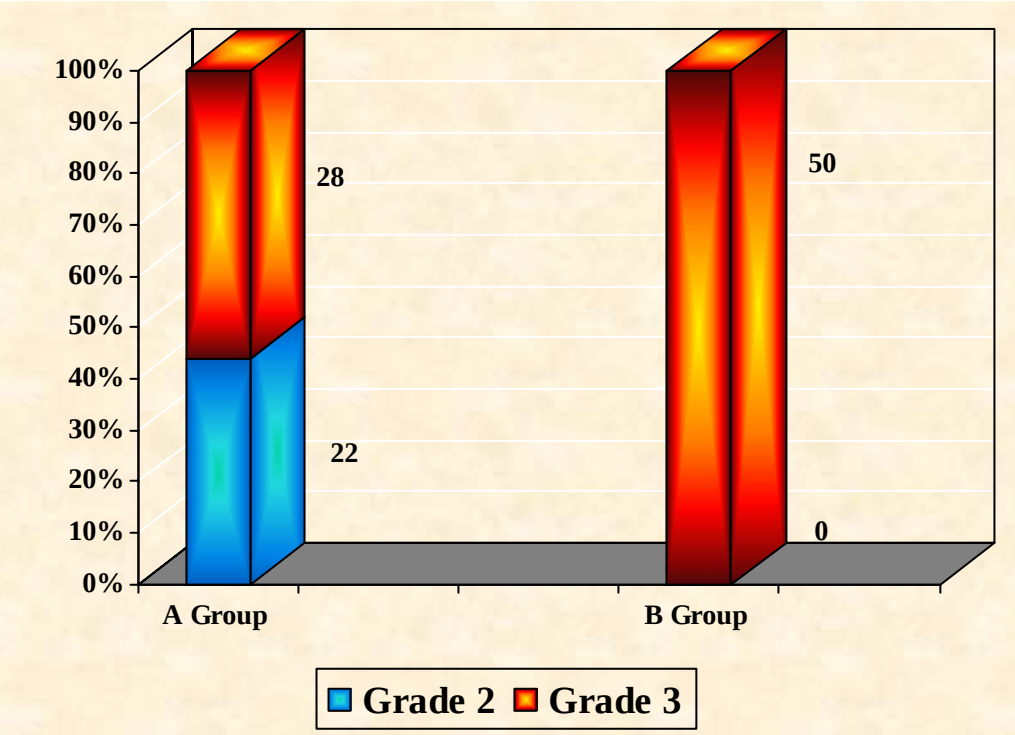

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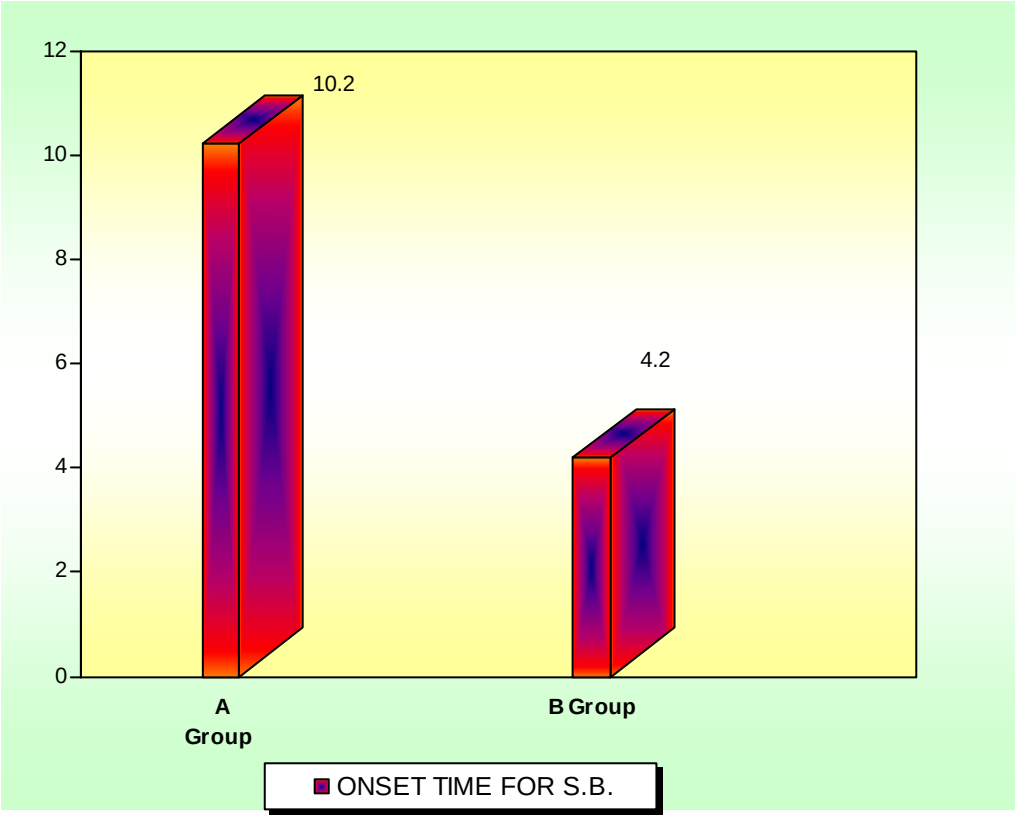

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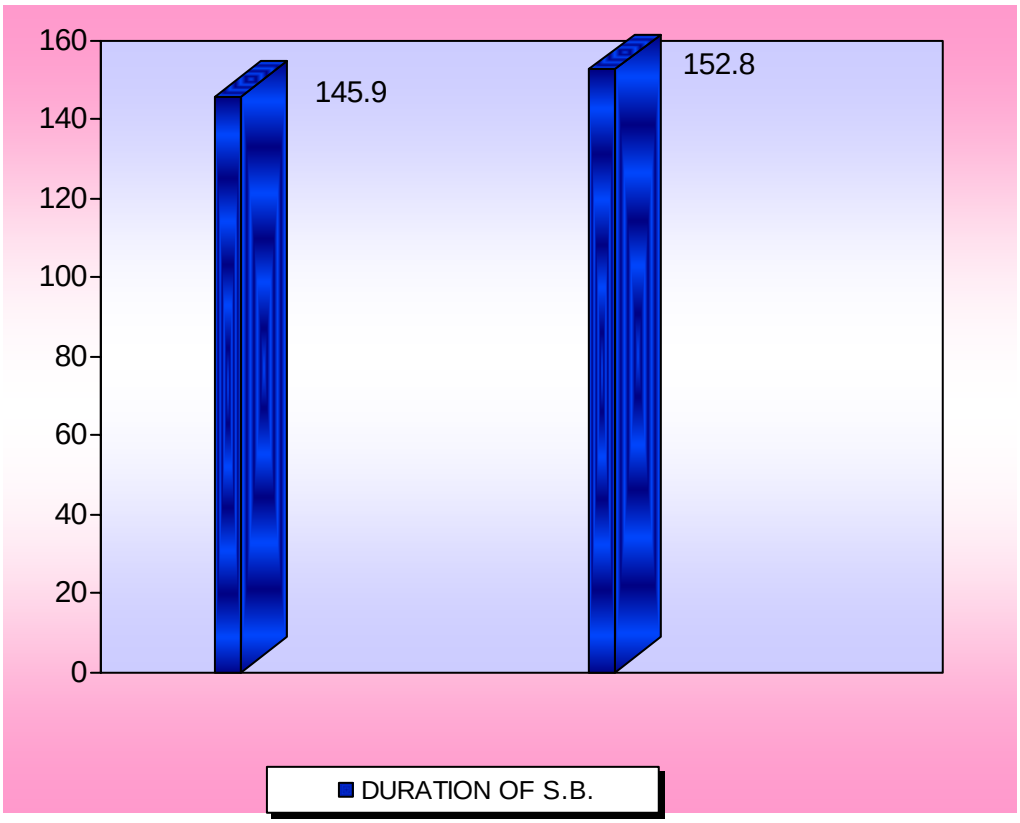

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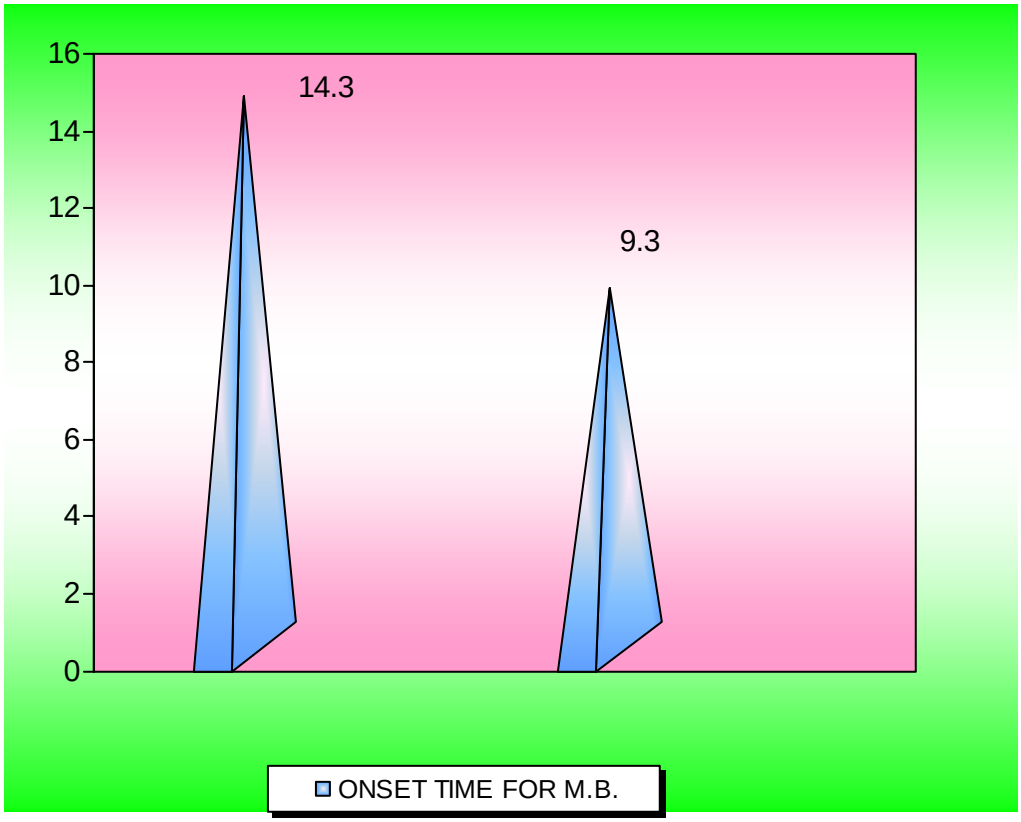

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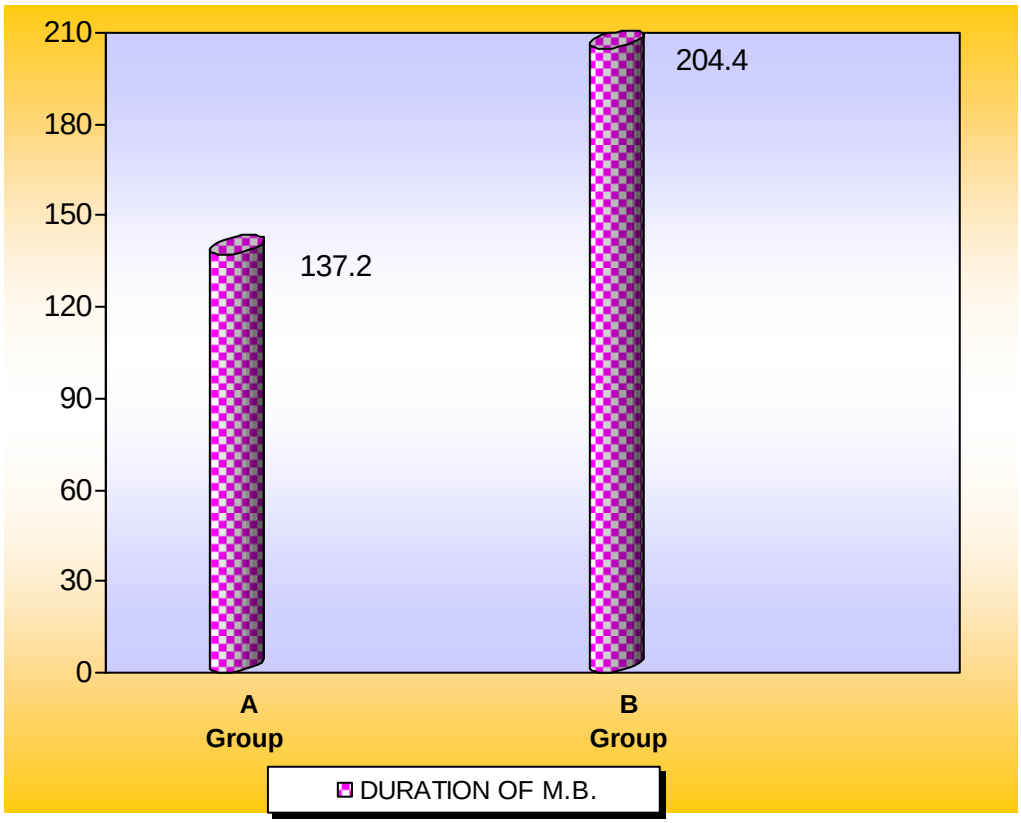

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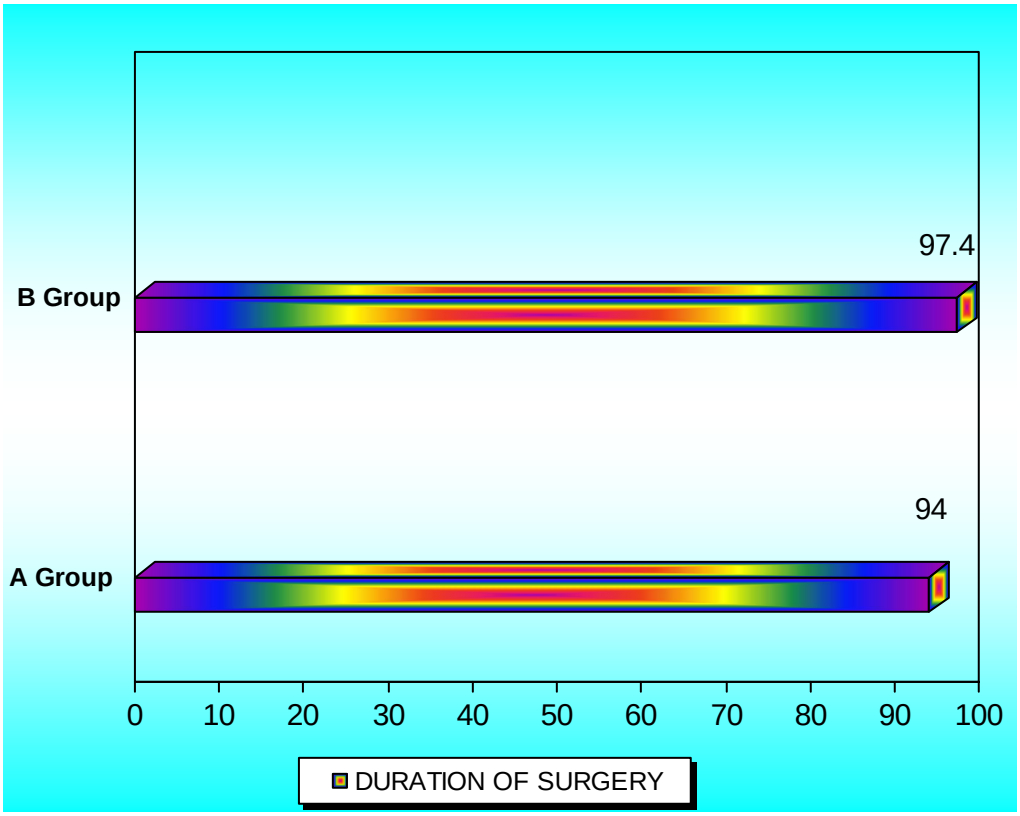

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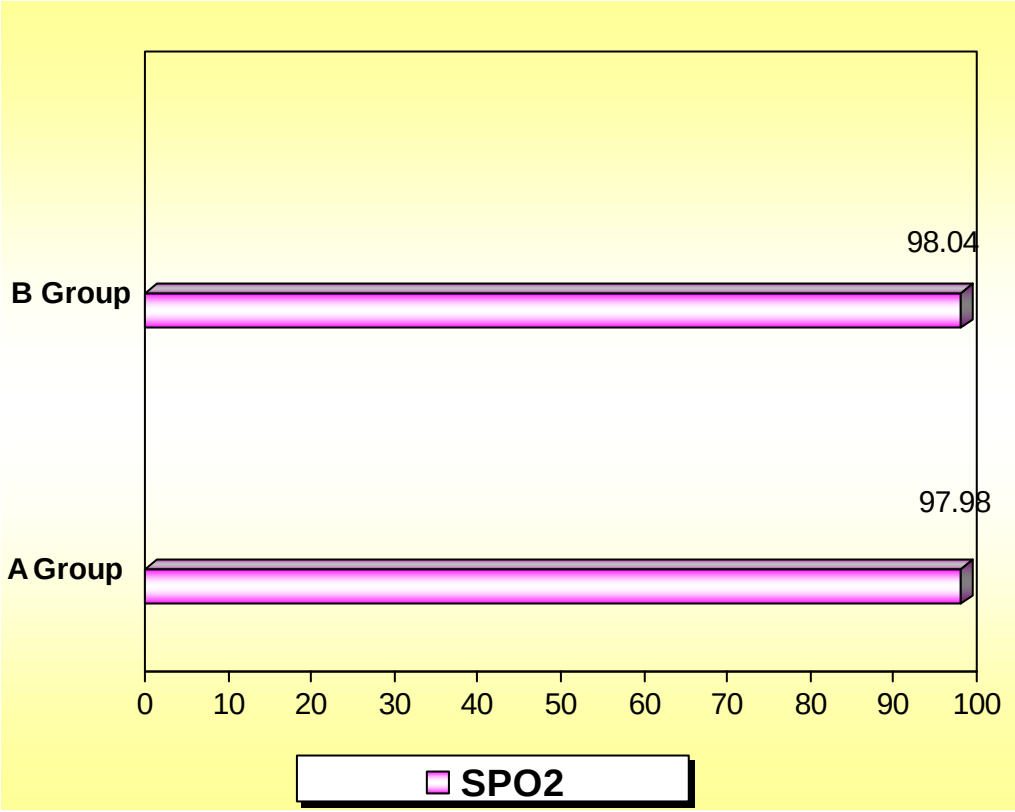

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